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THE INFLUENCE OF THE MIDBRAIN TRANSECTION AND PHENOBARBITAL ADMINISTRATION ON THE EXPERIMENTAL TRAUMATIC COMA

by

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I. INTRODUCTION

A transient reversible disorder of consciousness immediately following head injury is called cerebral concussion. Accompanying signs of cerebral concussion are paleness of skin, cold sweating, hypothermia or change of respiratory and circulatory functions simultaneously. Of all the phenomena due to head injury, the most important is the reversibleness of functional disorders in the nervous system.

There are three hypotheses concerning the etiology of cerebral concussion.

1) Transient anoxia or hypoxia in the central nervous system resulting from cerebrovascular changes caused by head injuries.

2) Functional, reversible reaction of the central nervous system to mechanical stress which may be associated with no morphologic changes. This may be described as "molecular reaction of the nervous system".

3) The existence of organic change in the central nervous system caused by mechanical stress.

As to the location of lesions in the brain in case of cerebral concussion, there are two hypotheses.

a) The lesions in the nervous system brought on by mechanical stress are not localized in some specific part, but diffuse throughout the central nervous system (DENNY-BROWN & RUSSEL, 1941, RAND & COURVILLE, 1946).

b) In cerebral concussion, lesions may be localized mainly in the brain stem, though the influences spread secondarily to the cerebrum and spinal cord.

The latter theory is based upon the disturbance of consciousness from inflammation, neoplasm and operative procedures in the diencephalon or midbrain.

Recently there are many experiments affirming the latter theory, in which when an experimental localized lesion is made in the reticular formation or the surrounding tissue of the midbrain or medulla oblongata by various experimental methods, there may appear the transient disturbance of consciousness (MORISON & DEMPSEY, 1942, ISHII, 1944, PENFIELD & JASPER, 1947, MORUZZI & MAGOUN, 1949,

MAGOUN, 1950, 1952, JASPER et al., 1952, PENFIELD, 1952, HAYASHI, 1959, MATSUMURA, 1959).

Microscopic hemorrhage in the brain tissue due to mechanical stress may be important in the discussion of the etiology of cerebral concussion. It had been shown by OGOSHI (1948), KUDO (1949) and others that there are hemorrhagic foci in cases of initial or secondary coma from head injury not only in the cerebrum but also in the midbrain, though not as frequent. In the present study, the author has studied the interrelation of the nervous function between cerebrum and brainstem in the case of experimental cerebral concussion in cats. First of all, air impact by means of air rifle discharge was given against the exposed dura overlying the parietal cortex in a cat whose midbrain had been transected intercollicularly already dy BREMER's method (1935). That is, the nervous connection between cephalic and caudal parts of the brain at the intercollicular plane was interrupted perfectly by making "*cerveau isolé*", and then air impact was given over the parietal region.

Secondly, phenobarbital was injected in an amount not enough to produce a change in clinical features, but enough to have some paralytic influence on the mesencephalic reticular formation, and then air impact was given in the same manner as in the first.

Thirdly, the experimental cats receiving preliminarily both midbrain transection and phenobarbital injection in the same manner as in the first and the second experiments were given air impact injury over the parietal region thereafter.

II. EXPERIMENTAL METHODS

1. Animals :

Unanesthetized adult male or female cats weighing from about 1.8kg to 4.2kg were used for experiments. Vagus nerve and carotid artery were maintained intact throughout the operation.

Inhalation ether narcosis by open drop method was used only initially for head-fixation, midbrain transection and insertion of electrode in the brain.

2. Procedure of the operation :

(1) General procedure :

Each cat was fixed on a hammock in prone position. In some cases the four limbs were allowed to hang down naturally.

The median skin incision of the head was done from nasal tip to occipital protuberance. Both temporal muscles were separated from the skull by blunt dissection.

In the occipital region was made a bone window which was long in the frontal plane and narrow in the sagittal plane. The posterior end of the window reached the tentorium. Then the dura was incised, exposing the occipital cortex of this area through the window in the skull.

The above procedures were preliminaries to midbrain transection. After making midbrain transection the skull window was filled and covered with temporal muscles

and skin. This procedure of opening and closing the skull window was done on all cats of all four groups of experiments.

(2) Method of midbrain transection :

The midbrain transection was done following BREMER. That is, through the skull window in the occipital region the occipital cortex was exposed and moistened with saline. Then the occipital lobe was pushed away anterocranially by means of a brain spatula inserted toward the tentorial incisura horizontally along the superior wall of tentorium. On reaching the tentorial incisura the cerebral spatula was raised to vertical and then the midbrain was transected intercollicularly throughout.

The ventral line of incision in the midbrain was terminated at the anterior limit of the pons, which was just behind the outlet of oculomotor nerve.

In order not to contuse the cerebral falx or GALEN's vein during midbrain transection, the bilateral transection was done separately.

Vascular injury of the ventral brainstem should be minimized and the basilar artery should be maintained intact. After midbrain transection the occipital skull window was repaired with temporal muscles and scalp.

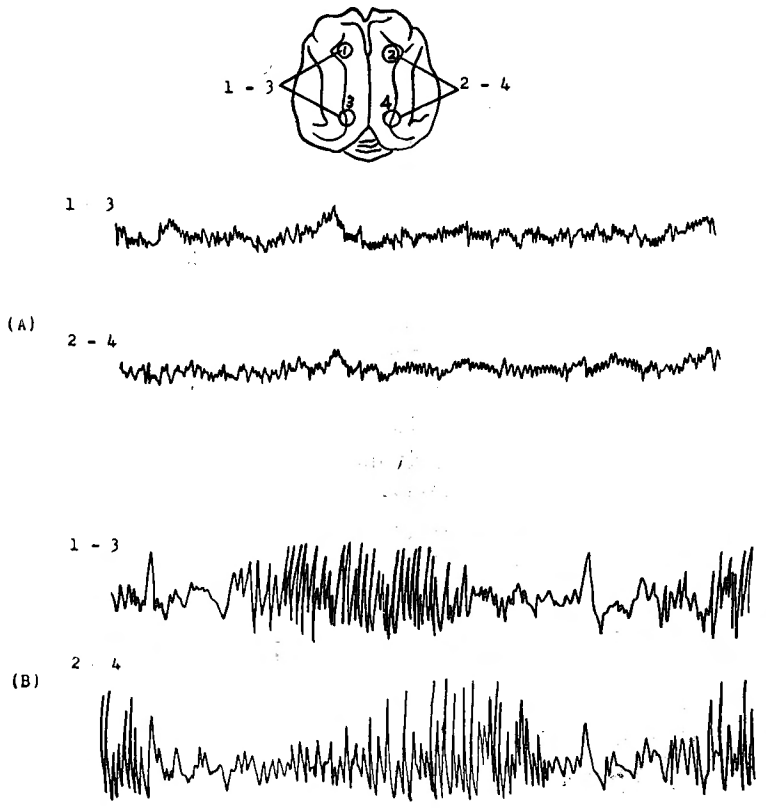


Fig. 1 EEG BEFORE AND AFTER THE MIDBRAIN-TRANSECTION
(Fronto-occipital bipolar leads)
A : Before Transection B : After Transection

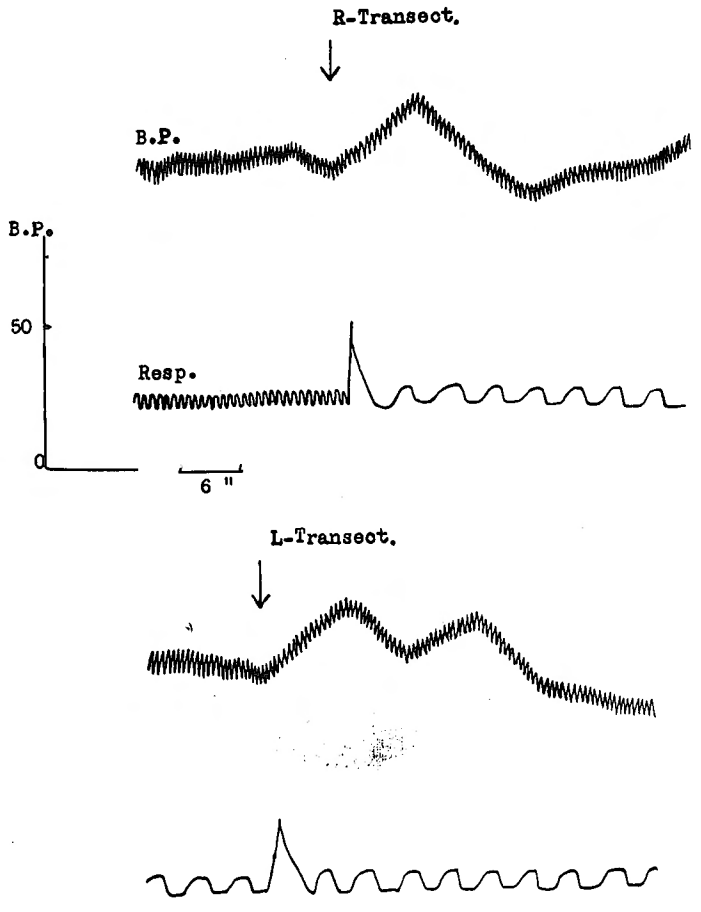


Fig. 2 CHANGE OF THE RESPIRATION AND BLOOD PRESSURE BEFORE AND AFTER THE MIDBRAIN-TRANSECTION

Whether the midbrain was perfectly transected or not could be known by the changes of EEG, clinical findings (particularly ocular signs) and postmortem dissection. There were the spindle burst waves of EEG, extreme myosis, immobility of the eye balls and fixed downward gaze. The light reflex of the pupils disappeared generally. There was generalized muscular rigidity, transient or permanent after midbrain transection. Following midbrain transection there were prominent changes in blood pressure and respiration ; that is, transient hypertension, bradycardia, spastic apnea, oligopnea or tachypnea (Fig. 1, Fig. 2).

(3) The method of phenobarbital injection :

Phenobarbital was dissolved in the solution provided directly before making injection in the lateral radial or ulnar cutanous vein slowly, for a total dose of 20 mg per kg.

(4) Insertion of the superficial and deep electrodes into the brain :

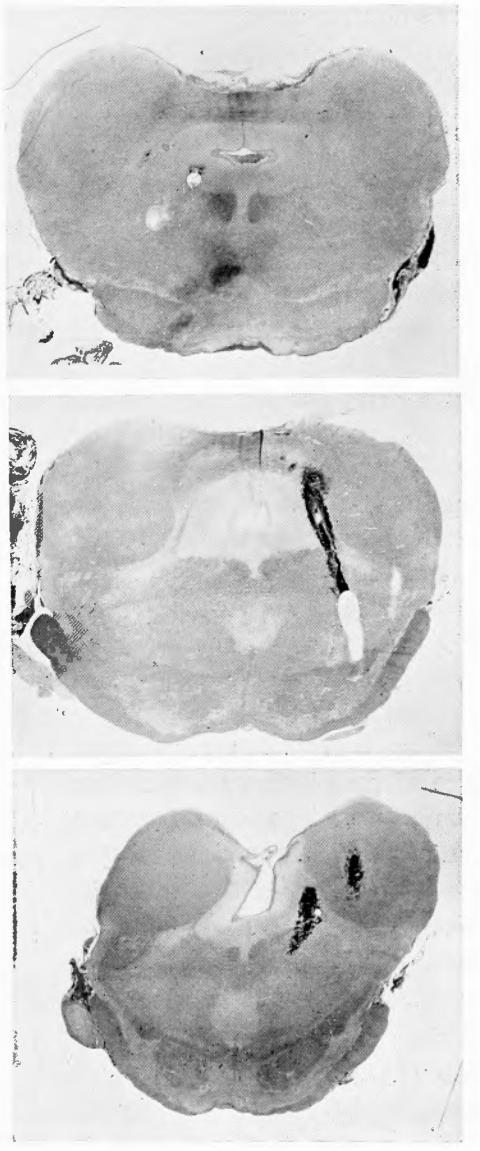
Lead electrodes for surface EEG consisted of four steel needles inserted to the

surface of dura at following four points through the skull which was widely freed of the temporal muscles.

a) Frontal point which is 5～10mm rostral from the coronal suture, 5～10mm lateral from the midline of the skull (right and left side).

b) Occipital point which is 5～10mm caudal from the coronal suture and 5～10mm lateral from the midline of the skull (right and left side).

Deep electrodes were made of stainless steel wire of about 0.2mm in diameter, each of which was insulated and fixed to each other by means of vinyl chloride



No.75

Transverse Section at the Level of 0.2mm. in front of the Interaural Line.

No.78

Transverse Section at the Level of 0.2mm. in front of the Interaural Line.

No.84

Transverse Section at the Level of 1.7mm. behind the Interaural Line.

Fig. 3 SITES OF THE SUBCORTICAL ELECTRODES (TRANSVERSE SECTION OF THE MIDBRAIN)

except for 2～3mm length at the tip. The bipolar electrodes were separated about 2～3mm from each other. When suitable craniectomy in the parietooccipital region had been done, the HORSLEY-CLARKE's stereotaxic instrument was placed on the head of the cats, and the bipolar electrode was inserted into the midbrain which was marked by GERARD's map at the point: 0.2mm, 4.0mm, 3.0mm, this being the reticular formation of the midbrain (GERARD et al., 1936).

After the experiments had been done the brain was taken out, fixed and subjected to KLUEVER-BARRERA's stain, then the insertion lesions of the deep electrodes were confirmed (Fig. 3). Surface and deep EEG were recorded with SAN-EI 2 channel portable electroencephalograph.

(5) Measurement of blood pressure and respiration :

Records were made on a kymograph by a mercury manometer for blood pressure, and by a balloon under a wide band fastened around the chest for respiration.

3. The method of the air impact :

A small hole about 6mm in diameter was made in the parietal region of the skull, 7～9mm lateral from the cranial midline. Then a steel pipe 10mm in length was inserted tightly into the small, well adapted parietal cranial hole. The muzzle of the air rifle was attached to this steel pipe tightly, then air impact was delivered on the exposed dura vertically (Fig. 4).

The energy of air impact force was about 9,800 erg. by 1 pump method, 29,400 erg. by 2 pump method on the average, which was measured by the potential energy of pendulum movement by delivering air impact on a certified weight hanging vertically. There appears a swelling of cerebrum out of the skull window of the occipital region following air impact, but the cerebral swelling may be minimized when the occipital skull window had been repaired well with temporal muscles and scalp. The cases in which the swelling or intracranial bleeding following air impact seemed marked were excluded in all experiments.

4. Criteria of the experimental coma :

In an animal the presence or absence of consciousness is a matter for conjecture. Therefore, the state of consciousness in experimental cats was judged indirectly according to nociceptive reflex and postural reflex (ARAKI, 1956). We should like to classify the general responsiveness of animals from the modification of GIRNDT (1932) and MATSUNAGA's (1959) criteria.

(1) Unresponsiveness of III degree or comatose state :

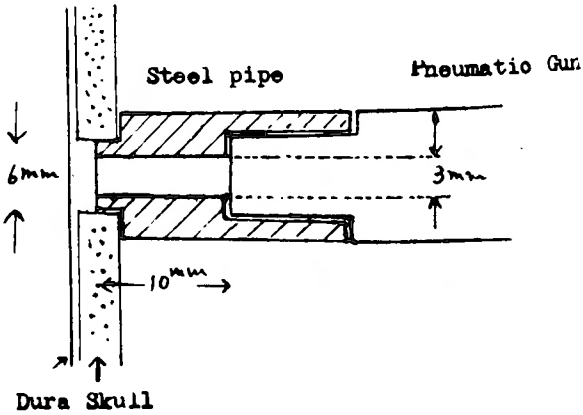


Fig. 4 DIAGRAMMATIC DRAWING OF THE AIR IMPACT APPARATUS USING A PNEUMATIC GUN

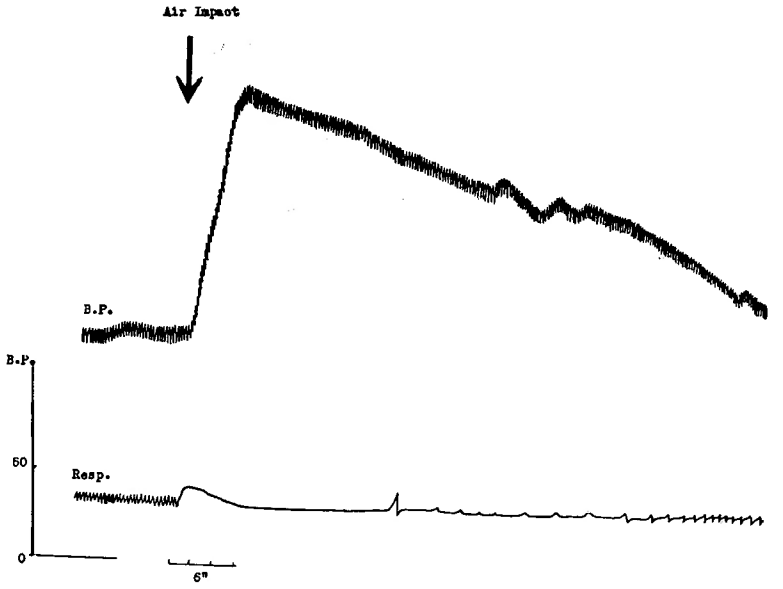


Fig. 5 CHANGE OF THE RESPIRATION AND BLOOD PRESSURE (GROUP I ; UNRESPONSIVENESS-III)

Nearly all of nociceptive reflexes or vital reflexes such as corneal, pinna reflexes, light reflexes are absent either completely or partially ; vomiting reflex by pharyngeal stimulus is lost, transient apnea, hyper- or hypotension, hyper and hypotonus of muscles are present (V & VI degree on GIRNDT's scale).

(2) Unresponsiveness of I and II degrees :

Postual reflex, spontaneous movement and response to smell stimuli are disturbed. Movements avoiding pressure or pain stimuli partially remain. There are moderate changes of respiration such as transient apnea on air impact (II-IV degree on GIRNDT's scale).

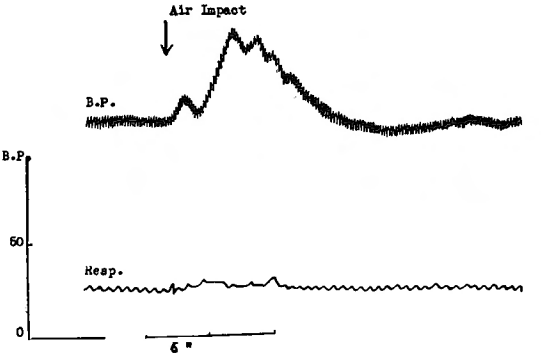


Fig. 6 CHANGE OF THE RESRIRATION AND BLOOD PRESSURE (GROUP I ; UNRESPONSIVENESS I-II)

(3) Intact responsiveness :

The animal shows no distinct difference in spontaneous movement, postural reflex and so on before and after the air impact (less than I degree on GIRNDT's scale).

III. EXPERIMENTS

1. The examination of the reaction in Group I (Control Group) which received no procedure but air impact after the occipital skull window was made, that is, the determination of the intensity of adequate air impact to produce coma in 100% of

Table Ia REFLEXES OR RESPONSES TESTED (GROUP I : 2 PUMP-METHOD)

		Respiration	Pulse	Pupils		Corneal Reflex	Nosetip[*3] or Ear lobe Reflex	Gag Reflex	Filght[*4] Reflex	Muscular Tonus of Extremities	Responsive-ness
				Size	Reaction to Light						
No. 38 ♂ 3.5 kg	A[*1]	100/m	150/m	norm.[*5]	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B[*2]	−(28″)	+	Mydr.[*6]→Myos.[*7]	−(20″)	−(25″)	−(24″)	−(30″)	−(30″)	hyper[*8]→norm.	
No. 56 ♂ 2.8 kg	A	50/m	120/m	norm.	+	+	+	+	+	norm	Unrespons. Ⅲ
	B	−(45″)	+	Mydr.→norm.		−(60″)	−(90″)	−(420″)	−(120″)	hyper.[*9]→hypo.	
No. 60 ♂ 3.2 kg	A	24/m	140/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(25″)	+	Mydr. ↓ Myos. ↓ norm.		−(30″)	−(20″)	−(40″)		hyper.→hypo.	
No. 61 ♂ 4.2 kg	A	30/m	180/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(65″)	+	mydr.		−(80″)	−(35″)	−(85″)		hyper.→norm.	
No. 66 ♀ 3.5 kg	A	150/m	180/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(17″)	+	mydr.		−(40″)	−(70″)	−(45″)		bypu.→norm.	
No. 74 ♀ 3.2 kg	A	66/m	150/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(60″)	+	myos.→norm.		−(240″)	−(210″)	−(80″)		hyper.→norm.	

[*1] A: Before Air Impact [*2] B: At Air Impact [*3] Nose tip Reflex: Flight reflex from pricking of nose tip
[*4] Flight Reflex: Flight reflex from pricking of forelimb [*5] norm.: normal in size or in tonus [*6] mydr.: mydriasis
[*7] myos.: myosis [*8] hyper.: hypertonus of muscles [*9] hypo.: hypotonus of muscles

Table Ib REFLEXES OR RESPONSES TESTED (GROUP I : 1 PUMP-METHOD)

		Respiration	Pulse.	Pupils: Size	Pupils: Reaction to Light	Corneal Reflex	Nosetip or Ear lobe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Responsiveness
No. 39 ♂ 4.0 kg	A	60/m	150um	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(50″)	+	mydr. ↓ myos. ↓ norm.		−(18″)	−(25″)	−(30″)		hyper.→norm.	
No. 52 ♂ 4.0 kg	A	21/m	180/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(10″)	+	mydr. ↓ myos. ↓ norm.		−(20″)	−(17″)			hyper.→norm.	
No. 75 ♂ 3.0 kg	A	180/m	180/m	norm.	+	+	+	+	+	norm.	Unrespons. I−Ⅱ
	B	−(10″)	+	mydr.→norm.	+	+	+	+	+	hyper.→norm.	
No. 84 ♂ 2.4 kg	A	180/m	180/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅲ
	B	−(20″)	+	mydr.→norm.	+	−(30″)	−(25″)	−(30″)		hyper.→norm.	
No. 92 ♂ 3.5 kg	A	120/m	180/m	norm.	+	+	+	+	+	norm.	Intact Response
	B	+	+	norm.	+	+	+	+	+	hyper.→norm.	

animals :

When the air impact was done by means of 2 pump air method for makng cerebral concussion, the cat's reaction was found to be unresponsiveness III (coma) in all cases, which was accompanied by transient hypertension and apnea (Tab. Ia, Fig. 5).

On the other hand, when the air impact was done by means of 1 pump air method, there reulted 3 cases of unresponsiveness III out of a total of 5 cats ; 1 case of unresponsiveness I-II, and another 1 case of intact responsiveness (Tab. Ib, Fig. 6). As shown in Fig. 6 (Unresponsiveness I-II) there was transient hypertension and dyspnea but the oligopnea was not as severe as in Fig. 5 (Unresponsiveness III).

In almost all cases, whether the experimental procedure was made by 1 or 2 pump method, there appeared a transient muscular hypertonia of extremities following air impact.

From the above it may be concluded that adequate stimulation to cause cerebral concussion by impact energy was more than 1 pump method and less than 2 pump method, so we have decide the 2 pump method as a sufficient intensity of stimulation for cerebral concussion.

2. Group II (The group of midbrain transection) :

The air impact was given by means of 2 pump method, after allowing a lapse of at least 30 minutes, usually 60 min. after the procedure of midbrain transection. The result was that the air impact which was of a strength adequate to produce coma in 100% of the animals in Group I, produced coma only in 4 cases (33%). In the cases of

Table IIa REFLEXES OR RESPONSES TESTED (GROUP II : UNRESPONSIVENESS III)

		Respiration	Pulse	Pupils: Size	Pupils: Reaction to Light	Corneal Reflex	Nose tip or Earl obe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Responsiveness
No. 22 야옹 2.2 kg	A*1	B*2 120/m	60/m	170/m norm. myos.	+	+	+	+	+	norm.	hyper.→norm.	Unrespons. III
	C*3	– (10″)	+	myos.	–	– (15″)	– (15″)	– (20″)	– (30″)	hyper.→norm.	
No. 24 야옹 3.0 kg	A	B	110/m	45/m	150/m 120/m norm. mydr.	+	+	+	+	+	norm.	hyper.→norm.	Unrespons. III
	C	– (10″)	+	mydr.	–	– (10″)	– (20″)	– (30″)	– (30″)	hyper.→hypo.	
No. 79 야옹 3.8 kg	A	B	180/m	48/m	220/m 180/m norm. myos.	+	–	+	+	+	+	norm.	hyper.→norm.	Unrespons. III
	C	– (15″)	+	mydr. → myos.	–		– (40″)	– (30″)	– (30″)	hyper.→norm.	
No. 95 야옹 2.9 kg	A	B	130/m	24/m	180/m 180/m norm. myos.	+	–	+	+	+	+	norm.	hyper.→norm.	Unrespons. III
	C	– (45″)	+	myos.	–	– (60″)	– (60″)	– (30″)	– (60″)	hyper.→norm.	

*1 A : Before Midbrain Transection *2 B : After Midbrain Transection *3 C : At Air Impact

Table II b REFLEXES OR RESPONSES TESTED (GROUP Ⅱ : UNRESPONSIVENESS Ⅰ-Ⅱ OR INTACT RESPONSE)

		Respiration	Pulse	Pupils: Size	Pupils: Reaction to Light	Corneal Reflex	Nose tip or Ear lobe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Responsiveness
No. 16 ♀ 3.0 kg	A | B	120/m | 45/m	200/m | 220/m	norm. | myos.	+ | −	+	+	+	+	norm. | hyper.→norm.	Intact Response
	C	30/m	160/m	myos.	−	+	+	+	+	norm.	
No. 77 ♂ 2.1 kg	A | B	160/m | 42/m	180/m | 210/m	norm. | norm. (right) myos. (left)	+ (right) | − (left)	+	+	+	+	norm. | hyper.	Unrespons. Ⅰ-Ⅱ
	C	−(20″)	150/m	myos. (right>left)	−	+	+	+	+	hyper.	
No. 81 ♀ 2.8 kg	A | B	160/m | 48/m	200/m | 180/m	norm. | mydr. ↓ norm.	+ | −	+	+	+	+	norm. | hyper.→norm.	Intact Response
	C	12/m	120/m	mydr.	−	+	+	+	+	hyper.→norm.	
No. 86 ♀ 2.4 kg	A | B	180/m | 30/m	180/m | 180/m	norm. | mydr.	+ | −	+	+	+	+	norm. | hyper.→norm.	Unrespons. Ⅰ-Ⅱ
	C	−(20″)	120/m	mydr.	−	+	+	+	+	norm.	
No. 88 ♀ 1.9 kg	A | B	100/m | 36/m	150/m | 140/m	norm. | mydr.	+ | −	+	+	+	+	norm. | hyper.→norm.	Intact Response
	C	24/m	170/m	norm.	−	+	+	+	+	hyper.→norm.	
No. 90 ♂ 3.0 kg	A | B	30/m | 24/m	180/m | 160/m	norm. | myos.	+ | −	+	+	+	+	norm. | hyper.	Unrespons. Ⅰ-Ⅱ
	C	−(20″)	140/m	myos.	−	+	+	+	+	hyper.	
No. 91 ♂ 3.5 kg	A | B	180/m | 36/m	180/m | 120/m	norm. | myos.	+ | −	+	+	+	+	norm. | hyper.→norm.	Intact Response
	C	36/m	180/m	myos.	−	+	+	+	+	norm.	
No. 93 ♂ 2.9 kg	A | B	150/m | 60/m	180/m | 70/m	norm. | myos.	+ | −	+	+	+	+	norm. | hyper.→norm.	Intact Response
	C	60/m	60/m	myos.	−	+	+	+	+	hyperno→rm.	

unresponsiveness III there appeared on one hand transient hypertension which proceeded to hypotension with bradycardia and on the other hand tonic apnea which proceeded to oligopnea (Fig. 7).

In the cases of intact responsiveness the blood pressure was increased or decreased but slightly. There was no change of the pulse pressure, but in some cases there was gasping respiration (Tab. IIa, IIb, Fig. 8).

3. Group III (The group receiving phenobarbital intravenously) :

About 20-30 min. after injecting a definite dose of phenobarbital intravenously there appeared spindle burst waves or high voltage slow waves in the EEG, but there were no clinical signs indicating sleep.

The air impact was delivered about 20～30 min. after taking phenobarbital. The unresponsiveness III was seen in 7 cases, unresponsiveness I-II in 2 cases, and intact responsiveness in 3 cases out of a total of 12 cases.

The influence on respiration and blood circulation resembled that in Group I (Tab. IIIa & IIIb).

4. Group IV (Intravenous phenobarbital plus midbrain transection) :

Phenobarbital injection was done about 30 min. after intercollicular

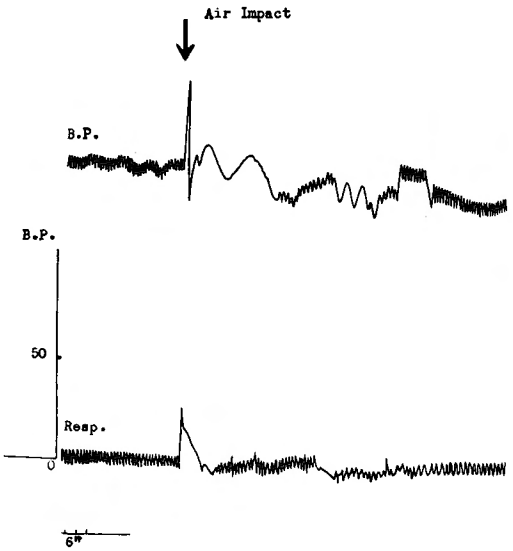


Fig. 7 CHANGE OF THE RESPIRATION AND BLOOD PRESSURE (GROUP II, UNRESPOSIVENESS III)

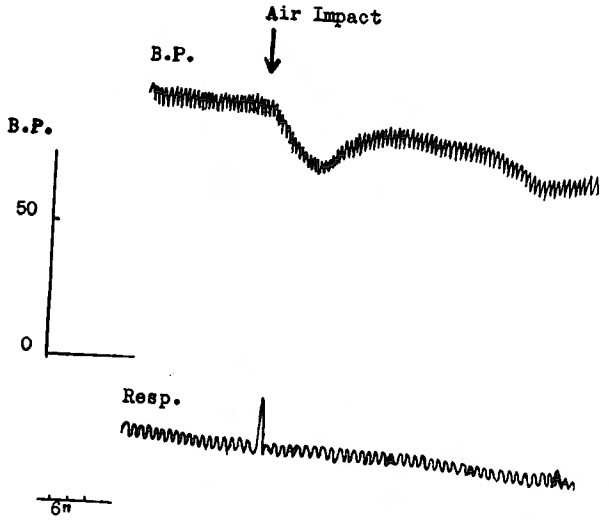


Fig. 8 CHANGE OF THE RESPIRATION AND BLOOD PRESSURE (GROUP II, INTACT RESPONSE)

Table IIIa REFLEXES OR RESPONSES TESTED (GROUP Ⅲ : UNRESPONSIVENESS Ⅲ)

		Respiration		Pulse		Pupils: Size	Pupils: Reaction to Light	Corneal Reflex	Nose tip or Ear lobe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Respon—siveness
No.42	A*1 B*2	120/m	150/m	150/m	120/m	norm	norm.	+	+	+	+	+	norm. norm.	Unrespons.
우 3.0kg	C*3	−(180″ artif.resp. 270″ spont.res.)		+		mydr. ↓ myos. ↓ norm.	− (90″)		− (200″)	− (240″)	− (300″)	hyper ↓ norm.	Ⅲ
No.43	A B	32/m	18/m	180/m	150/m	norm.	norm.	+	+	+	+	+	norm. norm.	Unrespons.
우 3.0kg	C	− (35″)		+		mydr. ↓ norm.			− (40″)	− (100″)	− (120″)	hyper. ↓ norm.	Ⅲ
No.44	A B	120/m	30/m	150/m	150/m	norm.	norm.	+	+	+	+	+	norm. norm.	Unrespons.
♂ 3.5kg	C	− (10″)		+		mydr. ↓ myos. ↓ norm.			− (13″)	− (13″)	− (15″)	hyper. ↓ norm.	Ⅲ
No.57	A B	30/m	24/m	150/m	150/m	norm.	norm.	+	+	+	+	+	norm. norm.	Unrespons.
우 2.2kg	C	− (45″)		+		mydr.			− (10″)			hyper. ↓ norm.	Ⅲ
No.71	A B	36/m	30/m	90/m	120/m	norm.	norm.	+	+	+	+	+	norm. norm.	Unrespons.
♂ 3.1kg	C	− (20″)		+		mydr. ↓ norm.		− (30″)	− (25″)	− (50″)	− (40″)	hyper. ↓ norm.	Ⅲ
No.85	A B	160/m	180/m	240/m	180/m	norm.	norm.	+	+	+	+	+	norm. norm.	Unrespons.
♂ 2.8kg	C	− (45″)		+		myos.		− (10″)	− (60″)	− (80″)	− (80″)	− (30″)	hyper. ↓ norm.	Ⅲ
No.89	A B	180/m	36/m	180/m	150/m	norm.	norm.	+	+	+	+	+	norm. norm.	Unrespons.
♂ 2.5kg	C	− (10″)		+		mydr. ↓ norm.		− (25″)	− (25″)	− (20″)		− (30″)	hyper. ↓ norm.	Ⅲ

*1 A : Before Phenobarbital Injection *2 B : After Phenobarbital Injection *3 C : At Air Impact

Table IIIb REFLEXES OR RESPONSES TESTED (GROUP Ⅲ : UNRESPONSIVENESS Ⅰ-Ⅱ OR INTACT RESPONSE)

		Respiration	Pulse	Pupils		Corneal Reflex	Nose tip or Ear lobe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Responsiveness
				Size	Reaction to Light						
No.41 ♂ 2.5kg	A | B	18/m | 20/m	120/m | 120/m	norm. | norm.	+ | +	+	+	+	+	norm. | norm.	Intact Response
	C	+(gasping)	+	norm.	+	+	+	+	+	hyper. ↓ norm.	
No.59 ♂ 2.5kg	A | B	120/m | 80/m	180/m | 180/m	norm. | norm.	+ | +	+	+	+	+	norm. | norm.	Unrespons. Ⅰ-Ⅱ
	C	-(30″)	+	norm.	+	+	+	+	+	hyper ↓ norm.	
No.67 ♀ 2.6kg	A | B	24/m | 24/m	150/m | 150/m	norm. | norm.	+ | +	+	+	+	+	norm. | norm.	Unrespons. Ⅰ-Ⅱ
	C	-(10″)	+	mydr. ↓ norm.	+	+	+	+	+	hyper. ↓ norm.	
No.72 ♂ 4.0kg	A | B	36/m | 36/m	120/m | 120/m	norm. | norm.	+ | +	+	+	+	+	norm. | norm.	Intact Response
	C	+(gasping)	+	myos. ↓ norm.	+	+	+	+	+	hyper. ↓ norm.	
No.78 ♀ 3.0kg	A | B	30/m | 150/m	150/m | 180/m	norm. | norm.	+ | +	+	+	+	+	norm. | norm.	Intact Response
	C	+(tachypnea)	+	mydr. ↓ norm.	+	+	+	+	+	hyper. ↓ norm.	

Table IVa REFLEXES OR RESPONSES TESTED (GROUP Ⅳ : UNRESPONSIVENESS Ⅲ)

		Respiration	Pulse	Pupils		Corneal Reflex	Nose tip or Ear lobe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Responsiveness
				Size	Reaction to Light						
No.65 우 3.1kg	A*1	60/m	180/m	norm.	+	+	+	+	+	norm.	Unresponsiveness Ⅲ
	B*2 C*3	50/m 48/m	60/m 180/m	myos. myos.	− −	+	+	+	+	hyper. → norm. norm.	
	D*4	−(70″)	+	mydr. → myos.	−	−(90″)	−(150″)	−(90″)	−(180″)	hyper. → norm.	
No.82 우 3.0kg	A	130/m	140/m	norm.	+	+	+	+	+	norm.	Unresponsiveness Ⅲ
	B C	60/m 42/m	120/m 180/m	myos. myos.	− −	+	+	+	+	hyper. → norm. norm.	
	D	−(40″)	+	mydr. → myos.	−	−(100″)	−(80″)	−(105″)	−(120″)	hyper. → norm.	
No.83 숭 3.2kg	A	54/m	180/m	norm.	+	+	+	+	+	norm.	Unresponsiveness Ⅲ
	B C	30/m 24/m	130/m 200/m	myos. myos.	− −	+	+	+	+	hyper. → norm. norm.	
	D	−(105″)	+	myos.	−	−(30″)	−(35″)	−(45″)	−(60″)	hyper. → norm.	
No.87 우 2.8kg	A	36/m	180/m	norm.	+	+	+	+	+	norm.	Unresponsiveness Ⅲ
	B C	15/m 15/m	210/m 210/m	mydr. mydr.	− −	+	+	+	+	hyper. → norm. norm.	
	D	−(60″)	+	mydr.	−	−(90″)	−(100″)	−(140″)	−(110″)	hyper. → norm.	

*1 A : Before Midbrain Transection *2 B : After Midbrain Transection *3 C : After Phenobarbital Injection
*4 D : At Air Impact

Table IVb REFLEXES OR RESPONSES TESTED (GROUPⅣ : UNRESPONS. Ⅰ-Ⅱ OR INTACT RESPONSE.)

		Respiration	Pulse	Pupils		Corneal Reflex	Nose tip or Ear lobe Reflex	Gag Reflex	Flight Reflex	Muscular Tonus	Responsiveness
				Size	Reaction to Light						
No.35 ♂ 1.8kg	A	30/m	180/m	norm.	+	+	+	+	+	norm.	Intact Response
	B / C	30/m / 25/m	150/m / 150/m	mydr. / mydr.	− / −	+	+	+	+	hyper. ↓ norm. / norm.	
	D	+	+	mydr.	−	+	+	+	+	hyper. ↓ norm.	
No.54 ♀ 2.4kg	A	120/m	26/m	norm.	+	+	+	+	+	norm.	Intact Response
	B / C	40/m / 40/m	30/m / 24/m	mydr. / mydr.	− / −	+	+	+	+	hyper. ↓ norm. / norm.	
	D	90/m	20/m	mydr. (right<left)	−	+	+	+	+	norm.	
No.55 ♀ 3.2kg	A	120/m	200/m	norm.	+	+	+	+	+	norm.	Intact Response
	B / C	30/m / 30/m	200/m / 180/m	mydr. ↓ myos. / myos. (right<left)	− / −	+(right) −(left)	+	+	+	hyper. ↓ norm. / norm.	
	D	30/m	150/m	myos. (right<left)	−	−	+	+	+	norm.	
No.63 ♀ 3.0kg	A	70/m	200/m	norm.	+	+	+	+	+	norm.	Intact Response
	B / C	30/m / 30/m	180/m / 180/m	myos. / myos.	− / −	+	+	+	+	hyper. ↓ norm. / norm.	
	D	180/m	130/m	myos.	−	+	+	+	+	norm.	
No.80 ♂ 3.4kg	A	190/m	120/m	norm.	+	+	+	+	+	norm.	Unrespons. Ⅰ-Ⅱ
	B / C	54/m / 24/m	140/m / 170/m	myos. / myos.	− / −	+	+	+	+	hyper. ↓ norm. / hypo.	
	D	−(5″) ↓ gasping	150/m	myos.	−	+	+	+	+	hypo.	

midbrain transection. Then, air impact was done 30 min. after injecting phenobarbital. There were no changes in clinical signs before or after injecting phenobarbital intravenously.

Muscular rigidity of extremities, immediately following midbrain transection intercollicularly, was observed transiently or permanently in all cases.

If the section of "*cerveau isolé*" was typical, myosis usually occurred, but in some cases mydriasis resulted instead of myosis. Perhaps this is because the superior

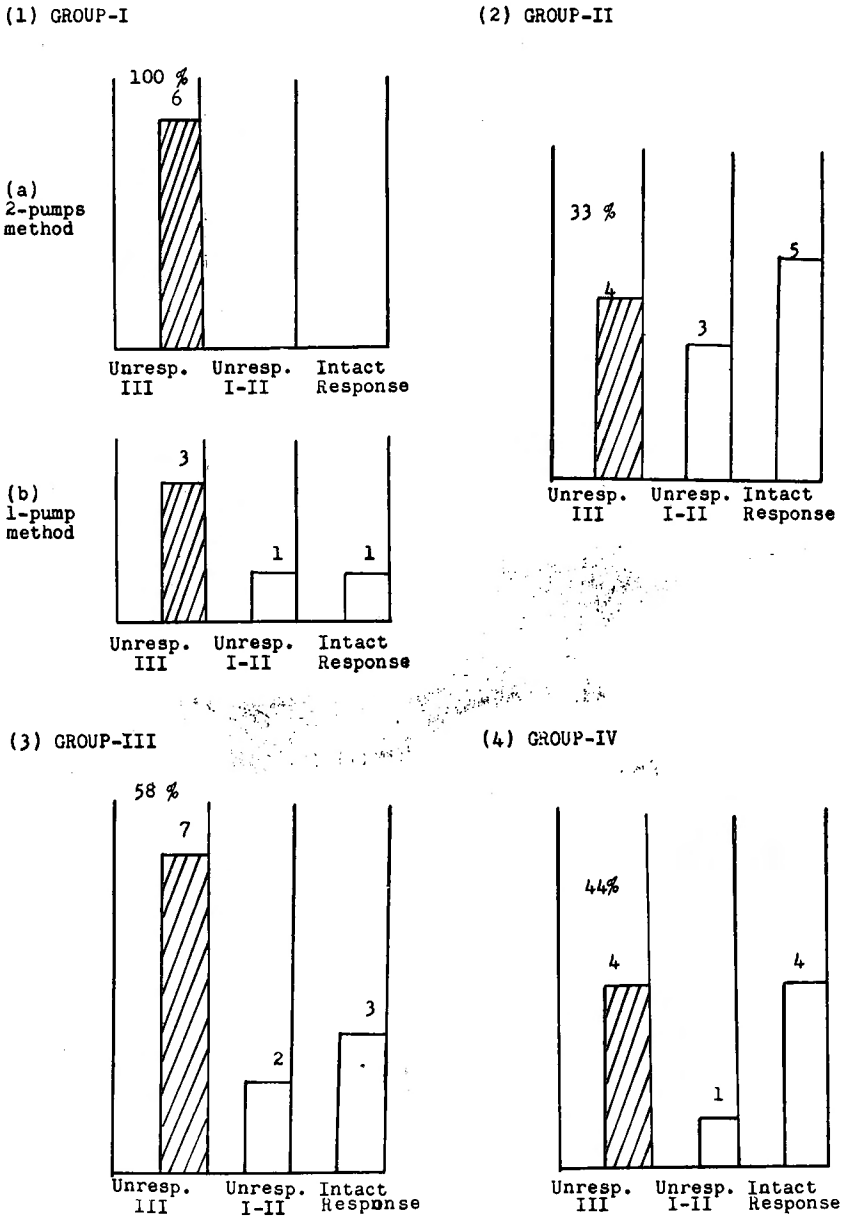


Fig. 9 PROBABILITY OF THE UNRESPONSIVENESS III BY MEANS OF THE AIR IMPACT

coliculus and oculomotor nerve tracts did not always remain intact.

In this group, unresponsiveness III resulted in 4 cases, unresponsiveness I-II in 1 case, and intact responsiveness in 4 cases out of 9 cases. The pupilary reaction following air impact might be transient myosis or permanent myosis after transient mydriasis (Tab. IVa, IVb, Fig. 9).

In the "*cerveau isolé*" cats there was occasionally absence of light reflex, or corneal reflex following air impact. Throughout all experimental groups, in cases of unresponsiveness III or I-II, respiration became gasping or hyperpneic associated with general excitability, and in some cases irregular oligopnea appeared following apnea after air impact.

5. Cerebral electrical changes in each experimental group :

(1) Group I :

a) No. 38 (Unresponsiveness III)

In this case air impact on the right parietal region has caused an artefact in all leads which lasts for 1～2 sec. Then low voltage and fast waves are present

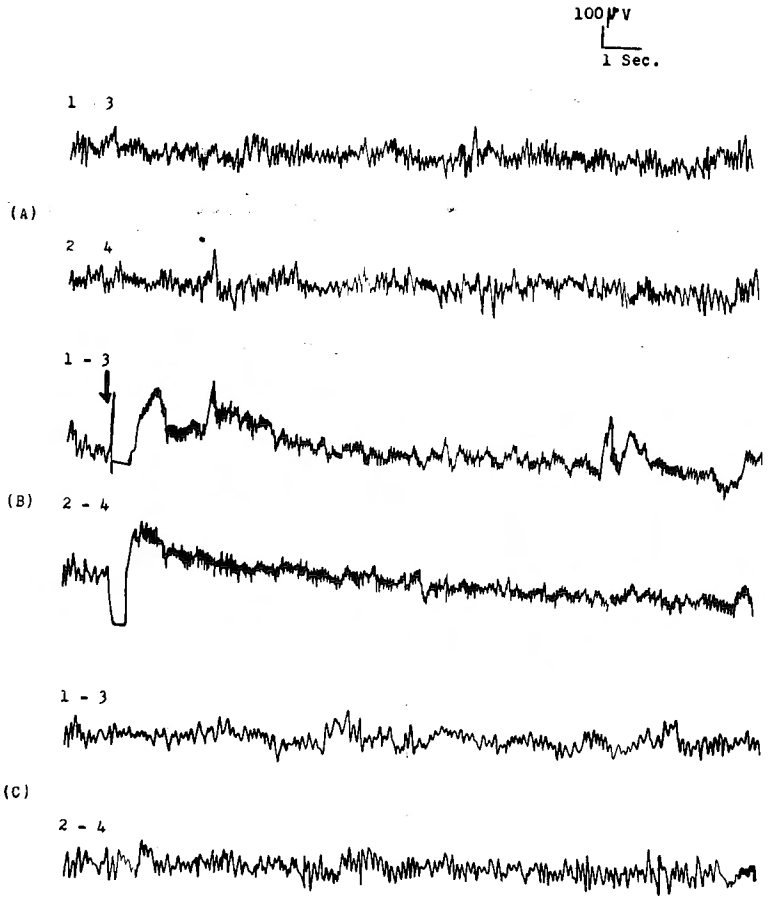


Fig. 10 EEG OF GROUP I, No. 38 (UNRESPONSIVENESS Ⅲ)
A : Before Impact B : At Air Impact C : 2min. after Impact

for about 30 sec. About 60 sec. following the end of the above changes, there appear high voltage and slow waves on EEG which may represent a recovery stage from cerebral concussion (Fig. 10).

b) No. 75 (Unresponsiveness I-II)

The electrical activity of the cortex and brain stem structure following the air impact are reduced in amplitude with little change in frequency, but at times essentially no change is produced. High voltage and slow waves of cerebral electrical potential are present 80 sec. later, which may represent a recovery stage from concussion (Denis Williams & D. Denny Brown, 1941). Changes in the electrical activity of brain stem structures largely parallel those in the cortex (Fig. 11).

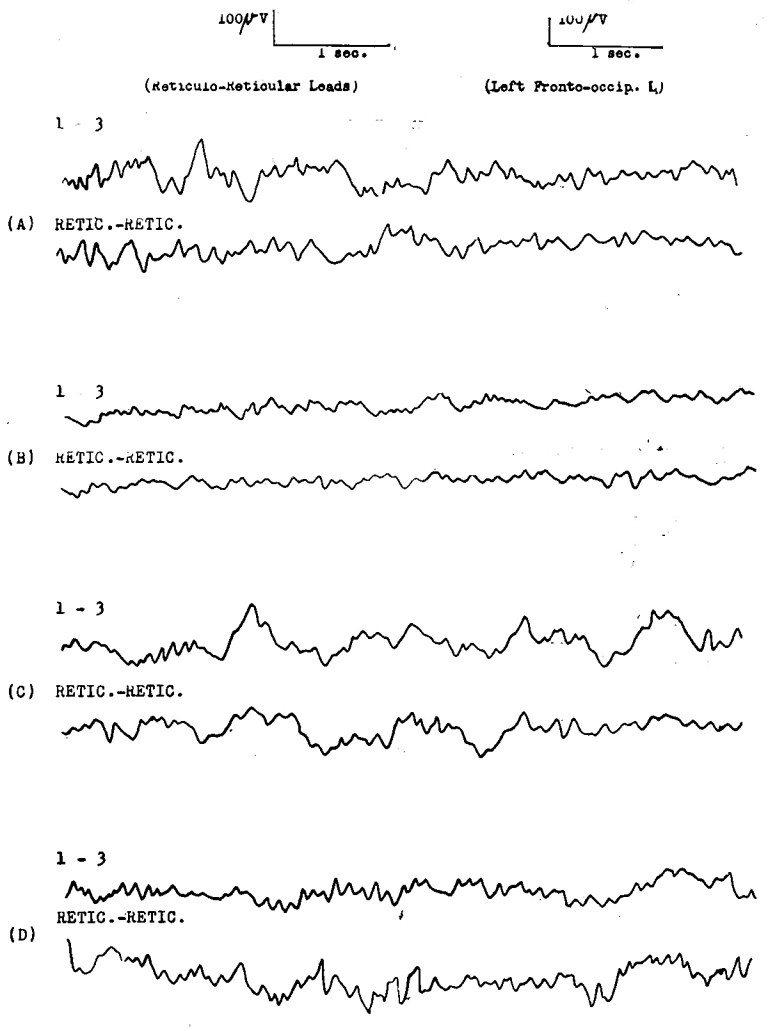


Fig. 11 EEG OF GROUP I, No. 75 (UNRESPONSIVENESS I-II)

A : Before Impact B : 10 Sec. after Impact
C : 80 Sec. after Impact D : 10 min. after Impact

c) No. 84 (Unresponsveness Ⅲ)

The findings of electrical activity of cortex and brain stem appearing immediately following air impact are generalized flattening. 20 min. later the electrical activity has almost returned to its former level, parallel with cortical and subcortical electrical changes (Fig. 12).

(2) Group II :

No. 95 (Unresponsiveness Ⅲ)

Directly following air impact, EEG shows generalized flattening and decrease in frequency. But the spindle burst waves which are present specifically in "*cerveau isolé*" preparations, are difficult to erase by air impact (Fig. 13).

(3) Group III :

a) No. 43 (Unresponsiveness Ⅲ)

Following air impact the EEG of the cortex shows flattening but the spindle

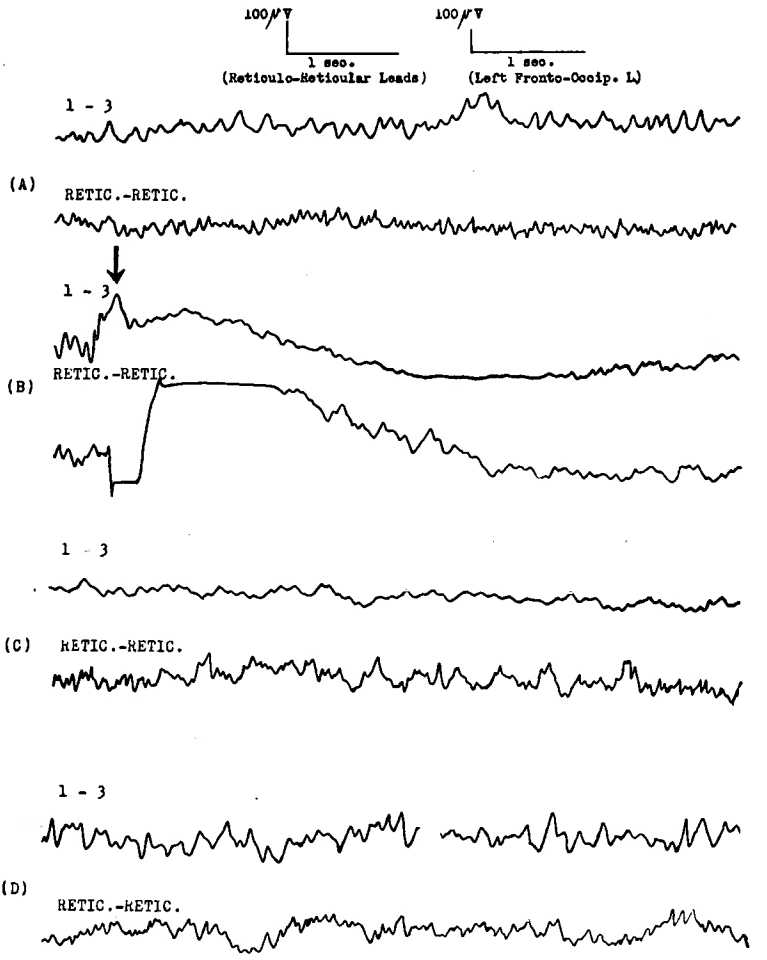


Fig. 12 EEG OF GROUP I, No. 84 (UNRESPONSIVENESS Ⅲ)
A : Before Impact C : 45 Sec. after Impact
B : At Air Imcact D : 20 min. after Impact

burst waves which were present after injecting phenobarbital intravenously, though decreasing somewhat in amplitude, remain for a long time (Fig. 14).

b) No. 67 (Unresponsiveness Ⅰ-Ⅱ)

Slow waves and spindle waves in the cortical EEG of this group show flattening following air impact. The subcortical electrical activity is seen also to be flattened but on recovery the later electrical activity shows greater depression than that of the cortex (Foltz et al., 1954) (Fig. 15).

(4) Group IV :

No. 63 (Intact responsiveness)

Following the combined preshot procedures, this group presents typical spindle waves in cortical electrical activity. The high voltage and low frequency waves

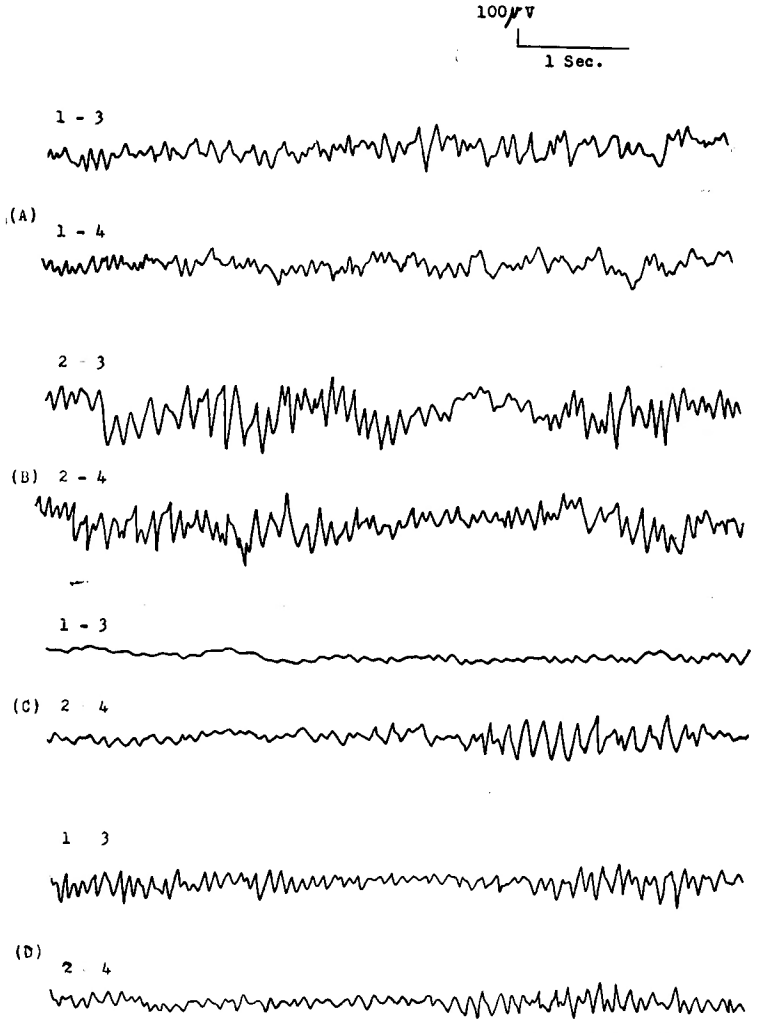


Fig. 13 EEG OF GROUP Ⅱ, No. 95 (UNRESPONSIVENESS Ⅲ)

A : Before Transection
B : 60 Sec. Midbrain Transection
C : 10 Sec. after Impact
D : 20 min.after Impact

in cortical EEG of Group IV are seen more clearly than in that experiment with phenobarbital injection alone. The spindle waves remain even following air impact, like in the cases of Group II. In this case the EEG does not return to the pattern before air impact, probably as a result of possible contusion of the occipital cortex under the skull window at the moment of air impact (Fig. 16). The severity of the cerebral electrical changes is usually not proportional to the objective signs of concussion (eg. loss of corneal reflex, respiratory paralysis and rise in blood pressure).

IV. DISCUSSION

1. Experimental production of cerebral concussion :

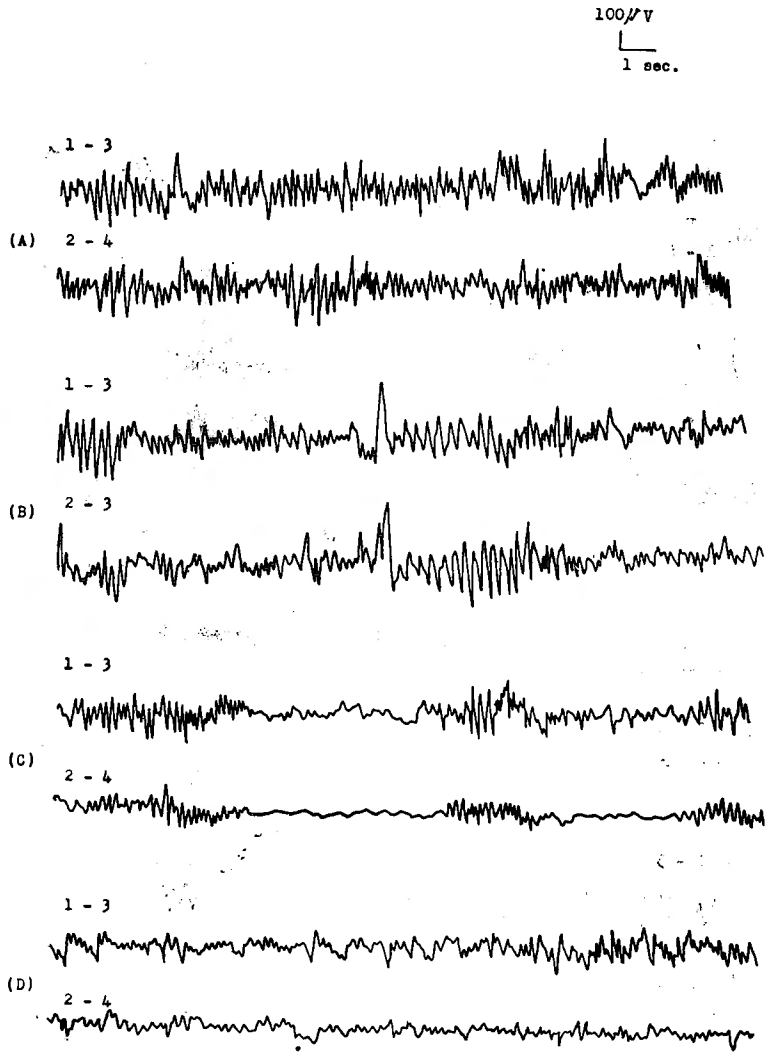


Fig. 14 EEG OF GROUP Ⅲ, No. 43 (UNRESPONSIVENESS Ⅲ)

A : Before Phenobarbital Injection
B : 20 min. after Phenobarbital Injection
C : 10 Sec. after Air Impact
D : 10 min. after Air Impact

The most important criterion for concussion is the occurrence of brief loss of consciousness immediately following a head injury. In addition disturbance of vital reflexes, and anomalies of respiration and circulation are noticed as somatic signs. In an animal, the presence or absence of consciousness is a matter for conjecture. The criteria for the existence of consciousness should be somatic signs; that is, disturbance of nociceptive and posture reflexes etc., or of respiration and circulation (GIRNDT, 1932, ARAKI, 1956) or EEG change (MEYER, DENNY- BROWN, 1955).

There are various methods of inducing experimental concussion, that is,

(1) Acceleration concussion (DENNY-BROWN & RUSSEL, 1941)

This is produced by a blow on the skull held loosely, an acceleration blow

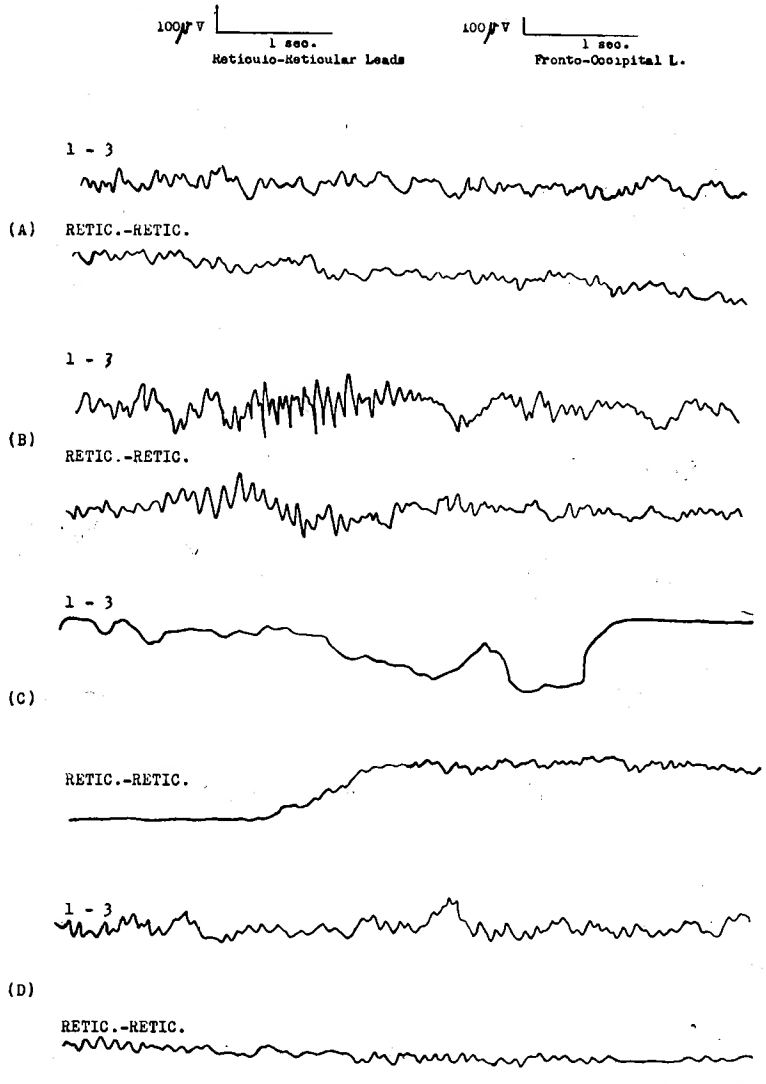


Fig. 15 EEG OF GROUP Ⅲ, No. 67 (UNRESPONSIVENESS Ⅰ-Ⅱ)

A : Before Phenobarbital Injection
B : 20 min. after Phenobarbital Injection
C : 10 sec. after Air Impact
D : 10 min. after Air Impact

of 29 feet per second by pendulum etc. By this method concussion can be produced under the condition in which the intracerebral pressure is not so high, but the movement of the head inherent in this method is undesirable for recording.

(2) Percussion concussion (WALKER et al., 1944)

Percussion concussion is produced after the method described by WALKER et al. A weight is dropped into a column of saline on the exposed dura from a maximum height of 4 feet 6 inches and rapidly withdrawn by a light cord and pulley. The measured duration of the blow is 0.1 to 0.2 sec.

(3) Compression concussion (MEYER & DENNY-BROWN, 1955)

Compression concussion is produced by sharply striking the plunger of a syringe

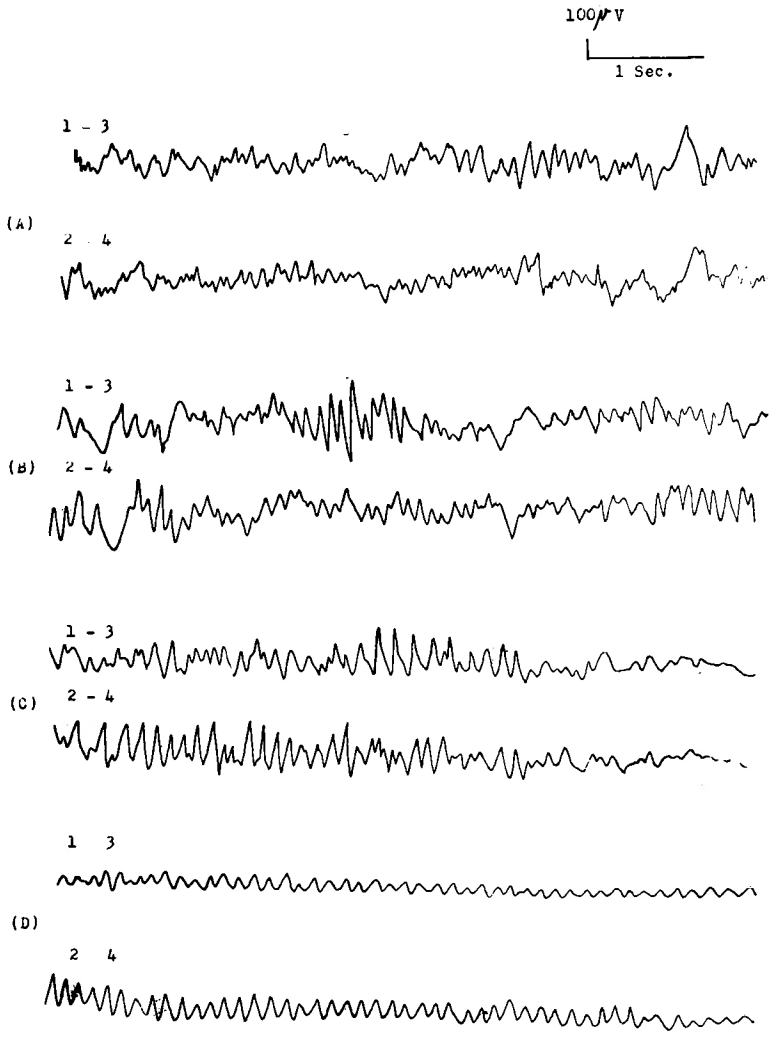


Fig 16 EEG OF Ⅳ, No. 63 (INTACT RESPONSIVENESS)

A : Before midbrain-transection
B : 1 min. after midbrain-transection
C : 20 min. after Phenobarbital Injection
D : 3 min. after Air Impact

designed for this purpose. The syringe is of 2 cc capacity and filled with air or saline and screwed into a suitable trephine hole in the skull, which has an outlet of 1 cm diameter. In this method the concussive stimulus is applied for less than 1/10 of a second.

The concussive stimulus in the present experiments can be classified as "compression concussion". In this method cerebral concussion can be reproduced invariably with no macroscopic hemorrhage in the cerebrum, and with extemely low death rate (NAGASAKI, 1959).

In the author's experiments the occurrence of some macroscopic change in the cerebrum at the moment of air impact cannot be denied, because of the presence of an artificial skull window over the occipital region, and the possible protrusion of the brain through it due to the changes in intracranial pressures resulting.

However, the cerebral concussion discussed here may be functional, the neurologic disturbance being transient or reversible and may not be secondary to ischemia of local brain, metabolic disturbance or mechanical destruction of brain matter. Therefore, the cerebral concussion in this experiment should be considered as generalized reversible functional disturbance of the nervous system following mechanical force.

2. Physiological mechanism of traumatic cerebral concussion (biological reactivity against the trauma) :

Often with fairly mild blows, not sufficient, but near to concussion strength, the animal presents slowing of the heart beat and falling of blood pressure (Fig. 8). There may appear simultaneously oligopnea or conversely transient tachypnea with hypersalivation and vomiting. Such subconcussive effects may be explained in general as due to the stimulation of the vagoglossopharyngeal mechanism. If in the same animal the vagi are sectioned, the respiratory, circulatory or other vegetative nervous changes disappear, a fact which indicates that those phenomena are based on the stimulation of such vagoglossopharyngeal mechanisms (DENNY-BROWN & RUSSELL, 1941).

In the EEG can be demonstrated desynchronization or synchronization. But we cannot clear the relationship between the concussive or subconcussive stimulus and the desynchronization or synchronization of EEG as a result of air impact.

In general the traumatic initial shock may be explained as the phenomenon of transient reversible disorder of the brain stem function due to head trauma. However, opinions on the mechanism of a transient reversible change of consciousness are still divided. The one is a transient neuronal paralysis arising from the brain trauma, the other is the result of excessive stimulation of the central nervous system at the moment of the blow to the head.

The latter opinion may be based on the following phenomena associated with cerebral concussion. That is :

(1) An immediate generalized muscular spasm spoken of as the tetanic stage of concussion which may be a result of intensive stimulation of the nervous function.

(2) The alterations of respiration consisting of a gasp of short duration with

resumption of normal respiration, transient irregular respiration or very transient or prolonged apnea. These phenomena may be due to a spasm of the intercostal or diaphragmatic muscles.

(3) A transient rising blood pressure occurring immediately or a few seconds later may be caused by an intense stimulation of the vasomotor centers which leads to peripheral vasoconstriction.

(4) Bradycardia may appear as a result of vagal excitation.

(5) Reflex changes, hypo- or a-reflexia.

Furthermore the large potentials recorded by WALKER et al. (1944) with a voltage divider after a concussive blow may be explained as true neuronal excitation. But the cerebral concussion following air impact may be accounted for by the disruption of the nervous function as a result of an abnormal excitation rather than the mere paralysis of the nervous system (ARAKI, 1957).

On the other hand, cerebral concussion is explained as a direct transient paralysis of central or peripheral neurons (DENNY-BROWN & RUSSELL, 1944). The reversible flattening or synchronization of EEG, arising from cortical injury potential and the rise in cortical oxygen availability are all explained by a transient paralysis of neurons. The cortical injury potential during concussion shows a remarkable similarity to the change of the motor stimulation thresholds (SPIEGEL et al., 1947). That is, the metrazol convulsion threshold is temporarily raised immediately following concussion. From these data, the general principle is advanced that the immediate effect of brain trauma of concussion may be a transient paralysis of neurons (MEYER, DENNY-BROWN, 1955).

3. The localization of the lesions produced by head injury with concussion:

In our experiments, the reflex arcs as an indicator of the experimental cerebral concussion are chiefly localized in the brain stem, but it may be necessary to make clear whether the impact force affects directly the reflex arcs themselves or whether it exerts the initial influence on the upper central nervous system which may regulate the reflex arcs of the brain stem, then secondarily affecting the reflex arcs.

In the first place it has been found that phenobarbital has a selective inhibitory effect on the reticular formation of the midbrain (ITO, 1958). Then in Group III, which had intravenous phenobarbital premedication, the cerebral concussion (Unresponsiveness III) following air impact is more difficult to produce than in the case of the control group.

Secondly, there are many studies showing the occasional occurrence of transient coma with some somatic symptoms following many kinds of stimuli in areas of the brainstem not directly involving reflex arcs, especially in the mesencephalic grey matter or reticular formation of the midbrain (ISHII, 1944, TAKETOMO & TODA, 1950, YABUNO, 1954, MATSUNAGA, 1959).

In the third place the functional disturbance of the nervous system in cerebral concussion following head injury is not localized only in the upper central nervous system but also extends to spinal reflex arcs. Furthermore the influence on spinal reflex function in cerebral concussion varies before and after cervical spinal transec-

tion (WALKER, 1944).

In the fourth place GROAT et al. have found that in experimental cerebral concussion sufficient to abolish the corneal or other reflexes, threshold in motor effect is raised momentarily to motor nuclei stimulus and for long periods to supranuclear pathways stimulus, but the threshold in motor effect to peripheral motor fibers stimulus does not change (GROAT et al., 1944).

Thus it may be possible to assume that the air impact force affects the brain stem containing each reflex arc or nucleus not only primarily by direct mechanical action but also secondarily by downward spread of some physiological action from the cortex to the brainstem reflex arcs.

In our experiment (Group II) some cases of decerebrated preparation can show coma (Unresponsiveness Ⅲ) following percussion concussion. But since in this group cerebral concussion after air impact can be seen less frequently than in the case of nontransected animals, the supposition of some descending depressive effect from the cerebrum to the brain stem may have to be admitted.

4. Unresponsiveness in Group II :

The air impact sufficient to produce coma in 100% in the group I animals (control group) caused coma in only 33% of the 12 cats in Group II. That is, by breaking the neuro-anatomical tracts between the cerebrum and brain stem in group II animals, the occurrence of coma can greatly be reduced after air impact.

EEG findings in "*cerveau isolé*" preparations show that the regular spindle burst waves with low voltage and slow waves persist even after cerebral concussion.

The results in group II may be explained by two possible factors.

(1) A descending suppressive impulse promoting the production of coma and transmitted from the cerebrum toward the brain stem.

(2) Transiently disturbed function of the brain stem caused directly by the impact force.

The above mentioned findings will agree with DENNY-BROWN's experimental findings following acceleration concussion (1941). They stated that in the decerebrated cat the intensity of the blow required to induce coma is higher than in the intact cat. They also stated that in localized percussion or compression concussion the effect on the brain stem is not dependent on the forebrain or on other parts of the brain but on the brain stem itself.

In our experiments this view must be revised, in that the descending suppressive influences from the forebrain are rather more important, because after midbrain transection air impact coma can surely be induced, but less frequently than in the intact (non-transected) cats.

In general, the muscular tonus of the experimental animals is increased after decerebration. This may be the result of neuroanatomical exclusion of the motor inhibitory center in forebrain and release of motor facilitatory center in mesencephalon, thus resulting in decerebrate rigidity.

On the other hand there are other factors involved in the hypertonicity of muscles. That is, the muscsular reciprocality between extensor and flexor, anesthesia

in the animal, level of the midbrain transection or intensity of the external stimulus, etc. (SPRAGUE et al., 1954, BACH, 1950, LINDSLEY & MAGOUN, 1948).

In the decerebrate animal the muscular tonus showed increase or decrease respectively following external stimuli applied to different portions of the midbrain. Furthermore a considerable influence is effected by decerebration on the function of vestibular nucles or cerebellum. The galvanic skin reflex after such mesencephalic transection becomes hypoactive. That is, the facilitatory center of galvanic skin reflex can be assumed to be localized only on the rostral side of the plane of midbrain transection (WANG, STAIN & BROWN, 1956).

The electrical activation of the cortical area takes place also in the midbrain transected animal in the same way as in the intact animal after stimulating the splanchnic nerve. This suggests that there is humoral activation of the cortex through the rostral portion of pontomesencephalic reticular formation (BONVALLET et DELL & HIBEL, 1954).

In general under midbrain transection the anomalies of the motor function, autonomic nervous function or cortical reactivity do not appear parallel to each other but vary diversely. In conclusion, the preserved activity of caudal mesencephalon in our experiment may be the result of interruption of the traumatic suppressive impulses from the cerebrum (descending suppression), which effect disturbed function of the brain stem.

5. Unresponsiveness in the Group III animals :

It has been reported that after injection of phenobarbital in an amount insufficient to produce a change in clinical features (sleep), stimulation of the mesencephalic reticular formation still shows the cortical arousal reaction, though somewhat hypoactive, and the recruiting reaction is strengthened. Therefore it may be concluded that even small doses of phenobarbital depress the reticular ascending system (ITO, 1958).

In group III coma takes place following air impact in fewer cases than in group I. Perhaps the reason for this may be as follows : Descending suppressive impulses from cerebrum toward the midbrain promoting the production of coma are weakened because of depression of the mesencephalic activating system due to phenobarbital.

6. Analogy of the neurological states between group II and group III :

"Cerveau isolé" (intercollicular transection) appears to cause the state of irreversible sleeping. Nevertheless in *"cerveau isolé"* preparation the olfactory and optic tracts and rostral portion of the mesencephalic reticular formation are still maintained, but the arousal reaction in EEG is hard to elicit (ARDUINI, A. & MORUZZI, G., 1952, BREMER, 1937, 1953).

The group III cats having phenobarbital in an amount insufficient to produce a change in the clinical features (sleep) show in the EEG the spindle bursts like those in group II (*"cerveau isole"*).

7. Unresponsiveness in Group IV :

This group had the combined procedures of both group II and the group III.

The coma on air impact may be expected to take place much less frequently, but in the present experiment it was not so infrequent as was expected (Fig. 9). For the definite explanation of this fact further study is needed.

V. SUMMARY

The author has studied the interrelation between the cerebrum and the brain stem of cats having experimental cerebral concussion.

An air impact was given on the parietal region of the intact brain in group I (control) and on that of the brain transected intercollicularly (*"cerveau isolé"*) in group II. In the group III cats, phenobarbital, which is said to have a paralytic influence on the mesencephalic reticular formation, was injected in an amount not enough to produce a change in the clinical signs, and then the air impact was given in the same manner. In group IV, the animals received both midbrain transection and phenobarbital injection and then the air impact was given. The strength of the air impact was the same throughout the experiment. Modified GIRNDT's classification was used as criteria for the disturbed consciousness, and the animals with changes severer than grade VI were judged as having the coma. In addition, blood pressure, respiration, and EEG were recorded simultaneously. The results of the experiment are as follows.

The air impact which was of a strength to produce coma in 100 % of the animals in group I (control), caused coma in only 33 % in group II (12 cats in all), 58 % in group III (12 cats in all), and 44 % in group IV (9 cats in all).

The animals receiving fairly mild blows, not sufficient, but near to concussion strength, presented signs of parasympathetic hyperactivity.

The mechanism of the coma due to the air impact may be the disruption of the nervous function caused by an abnormal excitation rather than the mere paralysis of the nervous function.

It might be assumed that the effect of air impact not only on the brain stem but also on the telencephalon and the diencephalon is of importance in the production of experimental coma.

Thus there seem to be two factors in the production of the experimental coma by means of air impact, 1) a descending abnormal suppressive impulse from the cerebrum toward the brain stem, 2) the disturbed function of the brain stem itself by transmission of a mechanical force.

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和文抄録

実験的外傷性昏睡に於ける中脳切断及び PHENOBARBITAL 投与の影響

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頭部外傷直後の一過性意識障害（無反応状態）が，脳の如何なる部分の，如何なる機能異常に起因するかについては，尚議論の多い所である．大別すると，外力に依つて，脳全体が障害を受け，その一過性神経異常が原因であるとする脳全体障害説と，脳幹局所がまづ障害をうけ，これが大脳乃至は脊髄にまで二次的に影響を及ぼすと考える脳幹障害説とに分けられる．又両者何れの場合でも，この機能異常が，単なる麻痺によつておこるものか，或は逆に機能の異常亢進の結果での無反応状態であるかについても明確に説明されていない．

著者は，空気衝激法に依つて惹起される，猫の実験的外傷性昏睡を分析する為に，種々なる前処置を行なつてその影響をみた．まづ，第1群の実験猫では，頭頂部に加えられた空気衝激で，全例に一過性昏睡が発現出来る最低衝激力を決定し，第2群では，Bremer の方法に随い中脳切断を行い，このあとで第1群と同一条件下で空気衝激を行なつた．即ち，第2群に於いては，前脳と脳幹との神経連絡をまづ機械的に遮断して後，外力を加えた．この為に外力は，脳幹に対しては，単に物理的刺戟として作用したものであり，少くとも外力による，前脳及び脳幹の間の神経生理学的作用の関与は除外出来たわけである．

第3群では，PHENOBARBITAL の少量即ち臨床症状には変化（睡眠）を来すに足りないが，脳幹網様織の機能低下を認め得る程度の量を投与後，第1群同様空気衝撃を加えた．

第4群としては，第2，第3群の両操作を，同一猫に加え，然る後第1群と同様条件下で空撃を加えた．

昏睡の指標としては，動物である為，意識障害を直接とりあげることは困難である．

随つて，脳幹レベルの変化の指標として，侵害乃至整位反射の異常，脳幹深部脳波を用い，呼吸血圧変化をも記録し，又，前脳レベルの変化に対して皮質表面脳波を記録した．

この結果，第2，第3，第4群は，第1群に比し，

昏睡発現率は有意の差を以て減少している．この説明として，昏睡発現に関与する2つの神経機能障害が想像出来た．

1） 空撃に依り，前脳から脳幹えと下降する，神経生理学的調整機能が障害される．この障害は結果からみると，昏睡発現を助長する．

2） たとえ空撃が頭頂部に加えられるとしても，この物理的外力は脳幹にまで波及するわけで，この結果脳幹局所の機能障害がおこり，昏睡を発現させる．

上記に説明される神経機能障害は，結果として，侵害，整位反射の消失等反射効果の抑制を示すものであるが，この抑制が神経機能麻痺に依るものか，逆に機能異常亢進によるかについては更に検討を要する．

次で第2，第3群の反応態度の類似性をも検討した．

結論として，脳振盪性昏睡は，1） 脳幹単独の局所性衝激でもおこり得る．然し，2） 頭部に加えられた機械的衝激によつて，脳幹よりも上位，即ち大脳レベルから脳幹に下降して，結果としては昏睡発現を助長するような，神経調整機能異常も決して無視することは出来ない．